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Case Study: Prevalence of Stroke in DKA

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Abstract
Diabetic Ketoacidosis (DKA) is a life threatening metabolic complication of Type 2 Diabetic Mellitus that often presents with neurological symptoms. A state of severe insulin deficiency, either absolute or relative, resulting in hyperglycemia and ketonemia. Although possibly underappreciated, upto 10% of cases of intracerebral complications associated with an episode of DKA, and / or its treatment are due to hemorrhage or ischemic brain infarction. Thrombotic risk during DKA is elevated by abnormalities in coagulation factors, platelets activation, blood volume and flow , and vascular reactivity.

Introduction

According to world health organization, diabetes mellitus is a chronic, metabolic disease characterized by elevated levels of blood glucose, which leads to damage to the heart, vessels, eyes, kidney and nerves. Over 90% of diabetes mellitus cases are T2DM. A condition marked by deficient insulin secretion by pancreatic islet beta cells, tissue insulin resistance and an inadequate compensatory insulin secretory response. Disease progression makes insulin secretions unable to maintain glucose homeostasis there by producing hyperglycemia.[1]

The organs involved in T2DM development include the pancreas, skeletal muscles, kidney, liver, brain, small intestine and adipose tissue. The evolved data suggest a role for adipokine dysregulation, inflammation and abnormalities in immune dysregulation, gut microbiota and inflammation have emerged as important pathophysiological factors.

Regarding the pathophysiology of diabetes mellitus, a malfunctioning of the feedback loops between insulin action and insulin secretions results in abnormally high glucose levels. In the case of beta cell dysfunction, secretion of insulin is reduce, which limits the bodies capacity to maintain normal physiological glucose levels. On the other hand insulin resistance leads to increased glucose production in the liver and decreased glucose uptake both in liver, muscle and adipose tissue. Even if both processes take place and contribute to development of the disease, beta cell dysfunction is more severe than IR. However, when both beta cell dysfunction and IR are present, hyperglycemia is amplified leading to the progression of Type II DM.[2]

DKA is a metabolic disorder characterized by hyperglycemia, ketonemia and academia due to severe insulin deficiency. It is also associated with a systemic inflammatory response characterized by vascular endothelial injury and coagulopathy. It is proposed that the oxidative stress induced by hyperglycemia and ketosis contributes to this inflammatory reaction and results in increased markers of inflammation, cytokines and

complement activation. Dehydration, hyperglycemia, hyperosmolarity, tissue hypoxia and acidemia – induced red blood cell rigidity which is a cause of hyperviscosity and vasoconstriction may all have an additive role regarding prothrombotic tendency. Despite is importance the exact mechanism by which DKA induces stroke still remains unclear. Relative insulin deficiency and subsequent increase in counter – regulatory hormones leads to increased gluconeogenesis and glycogenolysis and reduced uptake of glucose by peripheral tissues. Resulting severe hyperglycemia leads to osmotic diuresis and profound dehydration. Cerebral hypoperfusion followed by reperfusion injury correlates with the spectrum of cytotoxic and vasogenic edema is seen in patients due to DKA and is now hypothesized to be the mechanism of brain injury in DKA.[3]

Cerebral stroke is a medical emergency that can cause permanent neurological damage or even death. According to WHO, stroke was second most frequent cause of death worldwide in 2022 and the main cause disability.

Stroke is one of the major causes of morbidity and mortality worldwide and from being exceedingly harmful in diabetes, stroke is a disabling disorder. Classification of stoke into hemorrhagic and infarctive subtypes is bases on brain MRI, brain CT and WHO criteria

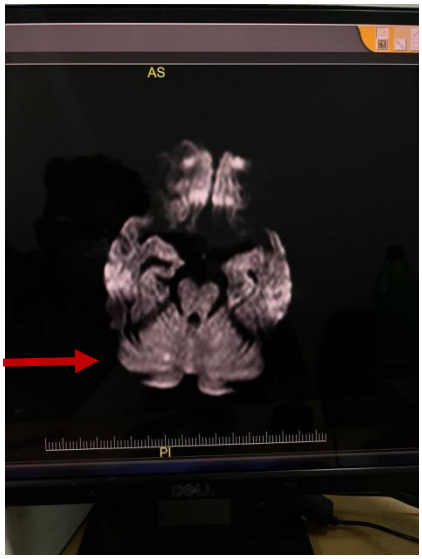
Here I am presenting a case demonstrating prevalence of stroke in DKA.

Case

An 81 years old female was referred to our Medicine department with history of drowsiness, decreased talk , dehydration and breathlessness for two days.

On examination, She appeared extremely dehydrated, lethargic, had dried lips and tongue with a loss of skin turgor. her weight was 55kg, height was 140cm, She was tachypnoeic with Kussmauls breathing. Her recorded respiratory rate was 30 breaths per minutes, Pulse was 120per minute, Blood Pressure was 120/75mmHg and axillary temperature was 36.8degree celsius.CNS: Coordination: finger to nose ☹ observed dysmetria and intention tremor): Pronation / supination – hand on thigh (observed intention tremor and a decrease in speed and accuracy on right side when performed unilaterally) toe tapping (negative), heel to knee (observed decrease in speed and accuracy), Range Of Motion (ROM) within normal limits at all major upper and lower extremity joints; Reflexes: bicep, tricep, patellar and archilles hypereflexia (grade 2) on the right side; Skin sensation: light touch, sharp-dull, temperature sensation al fully intact: Myotomes/ Dermatomes: normal; Limb matching test: unable to match right limb to left elbow and wrist. somewha consistently able to match right to left with hip, knee and ankle; Strength: upper and lower extremity grade 4 on isometric MMT or higher. ; Berg Balance Scale (BBS): score 36/ 56. Her estimated fluid deficit was 8% - 10%. The score in Glasgow Coma Scale was determined as 10.

Laboratory investigations showed ketonuria, acidosis (PH 7.29, PCO2 9.1mmHg PO2 135mmHg BICARB 9.9mmol/L ANION GAP 34.6) and hyperglycemia (plasma glucose 559mg/dl). Serum sodium concentration was found to be 132mmol/L, Potassium 3mmol/L. Level of HbA1C was 9.75% (normal range 4.8 – 6%) and urea nitrogen was 54mg/dl. Immediately maintenance and deficit replacement fluids were calculated. Administration of fluid deficit was planned within 36hours with a goal of 50% volume replacement within first 12 hours. Rehydration with 0.9% saline was started, followed by continuous intravenous infusion of a 5% glucose solution over 4 hours. Insulin infusion was started with 0.05U/kg/h. Capillary blood glucose was monitored hourly; electrolytes, urea and blood gases were repeated with an interval of 2 hours. In view of persistent low GCS, MRI brain screening showed acute infarct in right cerebellar hemisphere (figure 1).



MRI Brain of the patient shows an ischemic infarction of-right cerebellar hemisphere (marked by the arrow). (on DWI image)

To find the possible causes, screening for coagulation profile and prothrombotic conditions showed abnormal values. Blood culture yielded negative findings. Pus (bed sore) culture showed staphylococcus spp. Blood report showed neutrophilic leucocytosis with raised inflammatory markers (CRP: 222mg/L, ESR: 78mm/hr). Echocardiography showed no specific findings. She was started on intravenous antibiotics, antiplatelets, statins, closely monitored insulin therapy and other supportives.

One week later, the control cranial MRI showed unchanged lesions without progression. Rehabilitation therapy and supportive treatment were continued. Her neurological symtpoms gradually receded except for mild gait disturbance. After 20 days of hospitalization to select the right dose and timing of insulin therapy, she was discharged in good clinical and neurological recovery. A follow up cranial MRI and MRA were performed one month after discharge, showing siginificant improvement of the previously described area. Today, she can walk stably with completely regained neurological function and is in good metabolic control.

Discussion

[5]Diabetic ketoacidosis is a life threatening condition with high mortality and morbidity. This is predominantly assignable to intracerebral complications and results in death of 21% - 24% of affected patients. Several neurological deficiencies may be due to lack of recognition, ischemic – hemorrhagic stroke, an uncommon presentation during the DKA episode.

Cerebrovascular insult (CVI) is a known and important risk factor for the development of diabetic ketoacidosis (DKA): still, it seems that the prevalence of DKA among the patients suffering CVI and its influence of on stroke outcome might be underestimated. A cerebral hypoperfusion in untreated DKA may lead to cerebral injury, arterial ischemic stroke , cerebral venous thrombosis and hemorrhagic stroke[6]. These complicate the course and outcome of CVI. There is a considerable overlap of symptoms, signs and laboratory findings in the two conditions, making their interpretation difficult, particularly in elderly and less communicative patients[7]. Serum bicarbonate, pH, anion gap levels, blood gases should be routinely measures in all Type 2 Diabetes Mellitus , regardless of symptomatology , for early detection of existing or pending ketoacidosis. Stress hormone actions during the cerebrovascular incidents may precipitate DKA, but the reverse is also true. Diabetic ketoacidosis itself represents a risk factor for stroke, especially in adolescents. Hyprglycemia and acidosis may contribute to oxidative stress and lead to tissue ischemia. The risk of thrombosis during DKA is elevated due to the disorder of coagulation ,platelet activation , reduction of total volume and velocity of blood flow. There are serious limitations to the volume resuscitation rate to avoid rapid volume expansion and overload the cardiovascular system, the parallel and intensive use of osmotic solutions and loop diuretics may deep the existing dehydration level and aggrevate ketoacidosis itself.

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